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THE QUESTION OF EARLY OPERATION IN CASES OF INTRACRANIAL INJURY.

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IT is evident from cases and observations published from time to time, that the question of operative interference for intracranial injuries has not always been considered with due regard to the probable nature of the lesion. Since, in a large proportion of cases, unwarranted and ill-advised interference may lead directly to a fatal result, the most accurate obtainable knowledge possible of what is to be encountered in a prospective operation and the most exact appreciation of just what it may be expected to accomplish would seem to be essential; while in fact, a more or less prolonged period of unconsciousness has apparently been too often regarded as the sole and sufficient reason for entering the cranial cavity.

The value of a condition of unconsciousness, both as a diagnostic sign and as an indication for operation in certain cases, cannot be overestimated, provided the earlier history of the case can be obtained and verified. Unfortunately it often happens that such a history will be found to be entirely lacking, or to be dependent upon obviously unreliable evidence.

For example, in forty per cent. of a recent series of 50 cases, no information as to the primary mental state was obtained, and in many others it was known to the ambulance surgeon only from the unsubstantiated statements of casual witnesses.

The simple fact that unconsciousness exists, independent of the time of its occurrence and of its relation to concomitant symptoms, is in no sense diagnostic, since it occurs equally in any form of intracranial hæmorrhage—epidural, pial, cortical, or subcortical, or with a general cerebral contusion, with or without cerebral laceration.

The symptom of greatest importance to be studied in connection with unconsciousness, and which may be regarded as in some sort the key to its interpretation, is temperature. The writer is unable to better express his views as to the relation of temperature to unconsciousness, than by quoting from what he has written on a previous occasion.[1]

> The loss of consciousness, which immediately succeeds a cephalic injury, is always the result of diffuse cerebral contusion; if unconsciousness is preceded by a conscious interval, however brief, or if after restoration of consciousness its privation soon recurs, it is occasioned by some form of intracranial hæmorrhage. If, however, primary unconsciousness is permanent or greatly prolonged, its continuance may be due either to the severity of cerebral lesion or to a complicating hæmorrhage; and whether the one has persisted from the beginning or been at any time replaced by the other, or whether both exist together, can be determined, if at all, only by a study of all the symptoms presented. The pulse, temperature, and respiration must be systematically recorded in every case from the first opportunity afforded for observation until the end. . . . of these, the temperature in its course and variations will afford in the greater number of cases the most distinctive indication of the nature of the lesion. . . . If, then, after the lapse of hours consciousness still remains in abeyance, a stationary temperature, but one or two degrees above the normal standard, will indicate a hæmorrhage of some profusion without serious cerebral injury; but a higher elevation which constantly increases, with possible recessions, will point to a visceral lesion. . . . The cases in which consciousness after brief restoration is again lost permanently, or for a lengthened period, have the same relations to temperature as those in which unconsciousness has been uninterrupted. It will be recalled that the recurrence of unconsciousness after an early interval of sensibility is indicative of an increase or supervention of hæmorrhage, and that at a later period more or less conscious intervals in a general uncon-

scious condition result from a lessening from time to time of the hyperæmia or œdema of a diffuse cerebral contusion. The question of hæmorrhage should scarcely arise in the last instance, but the temperature still conforms to the general rule.

It is beyond the scope of this paper to enter into the detail of symptomatology except in so far as it may be essential to its main purpose—to indicate the surgical necessity of reaching, if not a positive, at least a reasonable conjectural diagnosis, and of being guided by it in determining the advisability of operative intervention.

The basic distinction to be made is between lesions as they occur above or below the dura mater. There ought to be no longer question as to the propriety of exploring fractures of the cranial vault by incision of the soft parts if they are not already compound, and by elevation of bone, if it be depressed. The recognition of a certain number of epidural hæmorrhages, is thus incidentally determined without the necessity of further consideration. If the fracture is merely linear, an epidural hæmorrhage, or other as yet undetermined lesion, must be relegated to the class of intracranial injuries in which the fracture is confined to the cranial base, or in which there is no cranial implication.

As hæmorrhage is the only epidural lesion so contusion in some form—meningeal or cerebral, limited or diffuse—may be said to be the only result of trauma, as it affects the structures below the dural membrane. Meningeal contusion occasions primarily hæmorrhages or serous effusions into or beneath the pia mater, and, secondarily, arachnitis, which by some surgeons has also been included within the sphere of operation. Cerebral contusion is manifested by hyperæmia, with thromboses of its minute vessels and capillary hæmorrhages, and by œdema. These several expressions of meningeal and cerebral injury may be limited or diffuse. General hyperæmia and œdema may be equally well marked as disclosed by post-mortem inspection, but oftener coëxist in inverse proportion. A superficial laceration with cortical hæmorrhage may be an accompaniment of cerebral contusion; or a secondary subcor-

tical laceration may result from fusion of capillary hæmorrhages, with gradual disintegration of tissue, or from a sudden and extensive destruction of cerebral areas due to ruptures of larger vessels, which have been undermined.

The subdivision of subdural lesions into meningeal and cerebral is important. The meningeal contusion is intermediate between the epidural and cerebral lesions, not only topographically, but in symptomatology and diagnosis; and if operation be proposed, in prognosis. The interstitial minute hæmorrhages and slight serous effusion may be neglected in this connection, since they are unrecognizable, and only material as they may lead to later local or general inflammation. The free hæmorrhage or serous effusion, however, which occupies the subarachnoid space may have direct symptoms which, if little complicated by those of concurrent injury of the brain itself, may be intelligible in exceptional cases. The hæmorrhage when too large to remain confined in the pial meshes, will still probably be in small amount and spread in a thin sheet, or in scattered patches, over some larger or smaller area of the pial surface, and complicated by cerebral lesions, which afford the dominant symptoms. In exceptional cases only will it be sufficiently large to produce paramount symptoms of hæmorrhage, or so concentrated as to occasion focal disturbances.

The frequency of occurrence of epidural hæmorrhage and of these other comparatively superficial lesions and the possibility of distinguishing them from each other, can only be estimated by statistical inquiry. In a series of personal observations of intracranial traumatisms first published in 1897,[2] it was found that among those subjected to post-mortem examination, 173 were of fractured base, or were without cranial implication. If to these are added twenty-seven others of the same classes from an unpublished series of 1904-5, the total of 200 will form a convenient and sufficient number for comparative study. Cases of gunshot and vertex fractures are excluded for obvious reasons.

In thirty-four of these cases epidural hæmorrhage was the dominant, or at least the most prominent lesion, as was pial

hæmorrhage in thirty-seven others. Epidural hæmorrhage occurred with fracture of the cranial base in 31 out of 148 cases, about twenty per cent.; and in 3 cases, somewhat less than six per cent., in 52 cases without such fracture. Pial hæmorrhage coëxisted with fracture of the cranial base in 21 out of 148 cases, a little more than fourteen per cent., and in 16 out of 52 cases, or nearly thirty-one per cent., of the cases of simple intracranial injury. If the case, therefore, be one in which cranial fracture may be excluded, it is probable in the proportion of six to one that the hæmorrhage is subdural, and if indications of cerebral lesion are not prominent that it is pial, rather than cortical.

The comparison of symptoms of these two forms of hæmorrhage, epidural and pial, will indicate statistical rather than pathognomonic differences. The opinion already expressed as to the impossibility of distinguishing one from the other is confirmed by such an examination.

The mental condition in the cases of epidural hæmorrhage varied: In 27, consciousness was lost at the earliest observation made: in 4 it was secondarily lost, and in 3 it was retained until the close of life. In 1 delirium was primary and permanent; and in another it followed a fifteen minutes' interval of primary consciousness. In the cases of pial hæmorrhage, consciousness was lost primarily in 28; and secondarily in 9, of which 2 were walking cases, and 2 were primarily delirious.

Both pupils were dilated in 7 of 30 cases of epidural hæmorrhage, and a single pupil was dilated in 11. In 5 cases one or both pupils were contracted; in 4 they were unsymmetrically abnormal, and in 3 they were normal. In the 11 cases in which a single pupil was dilated the hæmorrhage was on the same side of the cranial cavity in 9, as it was in all the 4 cases of unsymmetrical abnormality; it occupied the middle basic fossa of the same side in 3, and the opposite middle fossa in 2. In 27 cases of pial hæmorrhage, both pupils were dilated in 12, and a single pupil was dilated in 3; in 3 cases both pupils were contracted; in 1 case they were unsymmetrically abnormal; and in 7 they were normal. In the 3 cases in which a

single pupil was dilated, hæmorrhage was on the same side in all, but in 2 of them it was on both sides, as it was in the cases of unsymmetrical abnormality. In 2 cases in which hæmorrhage occupied a middle fossa, the pupils were normal in 1, and were both dilated in the other.

The pulse and respiration show no distinctive differences in the two forms of hæmorrhage, except that the average pulse was less frequent in pial hæmorrhage, on account, perhaps, of the more usual cerebral complication. The asymmetry of the radial pulse at the two wrists in a certain number of cases was common to all forms of intracranial injury. The respiration was not notably disturbed in either case until towards the close of life.

The focal symptoms of practical importance were disorders of the muscular system and occurred with equal frequency, whether or not the dural membrane intervened to protect the motor area. They were: paralyses, paresis, and local or general convulsions, muscular twitchings and rigidity. Some one or more of these were present in 22 cases, and with the exception of convulsions, occurred indifferently with either pial or epidural lesions. Convulsions were confined to cases of pial hæmorrhage, and in each instance were associated with cerebral laceration. The extreme infrequency of respiration noted in compression of the respiratory center by clot in pial or cortical hæmorrhage, and the cyanosis and pulmonary œdema which may characterize an epidural hæmorrhage involving a posterior basic fossa, are both focal and diagnostic symptoms, but without further significance, as they occur only in cases in which interference is not to be considered.

It follows from this view of symptoms that these two forms of hæmorrhage are indistinguishable from each other, while the cranial wall remains intact. Certain inferences may be derived from probabilities, the strongest of which is based upon the presence or absence of fracture, and the presence or absence of cerebral contusion. Consciousness is lost in the great majority of cases of either one; delirium is exceptional in either and is never occasioned by the hæmorrhage; pulse and respiration,

as well as focal symptoms, are not distinctive, the results of lumbar puncture are the same, and temperature in both depends upon complications. The pupils, both singly and symmetrically, are much more frequently dilated in pial than in epidural hæmorrhage; but so often in the latter that no useful inference can be drawn from their condition. In short, a hæmorrhage is a hæmorrhage wherever it is situated, and its location can only be indirectly established, if at all, whether it be pial, epidural, or cortical.

A subarachnoid serous effusion in considerable amount and distinguishable from an inflammatory product, usually general, but sometimes localized, was disclosed in fifteen cases. Cerebral lesions were well marked in all, as would seem to be inevitable since a meningeal contusion so severe could hardly fail to extend to the brain beneath. Direct symptoms might be expected to be recognizable only when the cerebral complication was comparatively unimportant, which was not the fact in either of these cases. Under what circumstances such a serous accumulation might reasonably be expected to exist may be better considered after some reference to the indications of brain injuries.

The symptoms referable to lesions of the brain itself, which in some aggregation characterize all cases of intracranial injury, may be briefly enumerated as disorders of the mind, pulse, temperature and respiration, muscular system, sensation, loss of urinary and fecal control, and occasionally disorders of the special senses. To these are to be added variations in the size and reactions of the pupils.

Intracranial injuries are always complex, but their component lesions usually differ so greatly in extent that only a single one may be of practical importance in diagnosis or in treatment. So many symptoms are common to two or more of these lesions that the determination of the one which is paramount must depend largely upon the study of symptoms collectively and in their mutual relations.

In this connection some points in symptomatology and diagnosis are pertinent to a later consideration of treatment.

The loss of consciousness is common to all forms of intracranial injury. It is, moreover, the only mental defect which directly results from either of the superficial hæmorrhages. Its relations to diagnosis in the matter of time of occurrence and temperatures, upon which its significance depends, have been already indicated; with hæmorrhages of importance it is almost, but not quite, an invariable symptom.

Delirium, which occurs in a limited number of cases in which hæmorrhage is a prominent lesion, is always, when present, a symptom of complicating superficial cerebral injury. Other less active mental disorders are also indicative of injury of a definite part and region of the cerebral substance.

Variations in size and reaction of one or both pupils are also common to all forms of intracranial injury. It was held by Hutchinson[3] that a single dilated pupil indicated a hæmorrhage in the middle fossa of the cranial base of the same side. Reference to the cases cited in the comparative study of pial and epidural hæmorrhages show that it may be observed as well in hæmorrhages occurring in other localities, and further reference to cases of cerebral lesion will show that it occurs in them independently of any hæmorrhage at all. The contention that it is a pathognomonic sign is thus fully controverted. There is no reason to doubt that, like other pupilary disturbances, it is the result of cerebral contusion; but in what relation they stand to specific cerebral injuries is undetermined.

Temperature probably affords the most important indication of the nature of the essential lesion. Its relation to unconsciousness has been elsewhere formulated. In general it may be said that, succeeding the possible subnormal temperature of shock in any form of injury its degree of elevation will measure the amount of injury to cerebral tissue. A moderately elevated early temperature, which is nearly stationary, indicates a superficial hæmorrhage with no more than the usual complicating cerebral implication. If, however, with reaction, temperature rises in a marked degree, cerebral contusion is to be regarded as an essential, if not the paramount lesion, and, if progressive, it may be assumed that structural cerebral changes

are also progressive, while remissions and exacerbations, aside from septic complications, indicate fluctuations in the amount of the characteristic hyperæmia and œdema. At a later period elevation of temperature may have other interpretations.

The pulse is not characteristic with hæmorrhages, but with a large proportion of cases of cerebral lesion its frequency affords a sharp contrast to the elevation of temperature, and may be considered of diagnostic value. The respiration both in hæmorrhages and contusions is often normal, and when abnormal is rather more frequently quickened than retarded. While in superficial hæmorrhages, it is often full, slow, and stertorous, it is not so with sufficient frequency to establish a general rule. As a positive sign it has some significance; as a negative indication, it has none at all.

Focal symptoms may furnish the most conclusive indication, not only of the site, but of the nature of the intracranial lesion. The most frequent of these are various impairments of the intellectual faculties in cases of laceration of the left prefrontal lobe. The fact that such disorders, aside from delirium, are connected with lesions of this origin alone has been adequately substantiated in the several publications of this writer at various times from 1894 to March of the present year. In one or two instances only, in which mental decadence had been manifested, there was found a large left frontal subarachnoid serous effusion without laceration; but even in these, as in all the others, there was sufficient evidence of contusion in hyperæmia and œdema if the case was recent, and of sclerosis if it was of longer standing.

Aphasia results from a cerebral lesion involving one or more of the speech centers situated in the third left frontal convolution and left temporal lobe. It has been asserted that it is also a result of compression of this region by a superficial hæmorrhage. The cases cited in support of this contention are not convincing, and are equally opposed to clinical and post-mortem observation, and to anatomical considerations.

Disorders of the muscular system occur in all forms of intracranial lesion, but not all of them with equal frequency

in each. Hemiplegia or hemiparesis, or paralysis affecting extremities of the opposite side, is not infrequent in cases of depressed fracture of the vertex, either from direct laceration of the motor area or from a superficial hæmorrhage; but with fracture of the cranial base, or with intracranial injury without fracture, it is probable that such a paralysis is the result of a hæmorrhage only, since cerebral laceration in this situation is unusual from contre-coup. Some degree of facial paralysis which is more frequently manifested than the other paralyses, and is sometimes due to injury to the facial nerve in its petrous position or to compression near its origin, is oftener a symptom of cerebral contusion, and perhaps explicable by gravitation of the serous fluid of an œdematous brain from above into the more dependent facial area. Paralyses of the ocular muscles are usually the result of lesions involving the corresponding cranial nerves at their origin. The same explanation is to be given of the paralysis occasionally observed of the pharyngeal muscles. General or local muscular twitching or rigidity are occasioned by the cortical irritation of hæmorrhages, or by direct superficial laceration. Convulsions on the contrary are not usual as a result of hæmorrhages, but indicate cerebral lesions of severity. Disorders of sensation are less frequent than paralyses, with which, if they occur, they are likely to coëxist.

The loss of rectal and vesical control, or of vesical control alone, which is the more frequent, is confined to cases of cerebral contusion of severity; and may be regarded as nearly pathognomonic. It occurs in a large proportion of both fatal and recovering cases of this character. It may be incontinence, but oftener the bowels move with some regularity; urine is discharged in considerable quantity and at more or less regular intervals. The sphincters are not paralyzed, and the lack of control is independent of the loss of consciousness, as it occurs equally when consciousness is retained. The explanation, therefore, must be sought in lesion of some specific centres of cerebral control as yet undetermined.

The occasional loss or impairment of one or more of the

special senses is due to mechanical injury of nerves in a line of fracture, to their compression by blood or other fluids in a basic fossa, or to lesions of a centre of control. Instances of the first are noted in deafness from implication of the auditory nerve in a fracture through the petrous portion of the temporal bone, and in blindness from implication of the optic nerve in a fracture through the optic foramen. Loss of the senses of taste and smell occurs and may be permanent in recovering cases, when, of course, the cause remains unknown. In some fatal cases with laceration of the frontal lobes the olfactory bulbs are found to be destroyed.

These varied symptoms of intracranial traumatism occur in manifold combinations according to the nature, severity, and complexity of lesions. When, as often happens, two or more lesions of nearly equal urgency or importance coëxist, with perhaps many symptoms in common and others ill-defined, or with a single one overshadowing all the rest, exact diagnosis may be impossible; but even then such knowledge of conditions present as may be attainable will ordinarily be sufficient to enable the surgeon to reach a reasonable conclusion as to the propriety of an operative interference.

In reaching such a conclusion several questions will demand consideration:

1. What may an operation be expected to accomplish?
2. Is it practicable?
3. Will it improve the patient's chances of recovery?

The answers to these several queries must be found in a fairly correct appreciation of the pathological conditions present. This will assume the recognition of the primary division of lesions into epidural and subdural as of essential importance.

The epidural lesion is easily accessible, and in suitable cases the operation for its relief involves a minimum danger and accomplishes a definite object. The subdural lesions on the contrary are in great part inaccessible, and the attempt to reach them by operative means is never without serious danger and always of doubtful expediency.

The operation for the removal of an epidural clot is in

itself practically, if not absolutely, without risk. The use of an anæsthetic is in a majority of cases uncalled for; sepsis and hæmostasis are within the control of the surgeon; and invasion of the cranial wall is unattended by serious shock. The object to be attained is simply the removal of pressure, and in the comparatively uncomplicated lesion and within the early period of time here contemplated, no irreparable nutritive changes will have occurred. The longer the cerebral compression is continued the stronger the probability that its effects, originally confined to hyperæmia and thromboses of the cerebral capillaries, will extend to, and fatally compromise the integrity of, the cellular elements. If it has been decided that a resort to operation is advisable, there should be no unnecessary delay after reaction has been established. If coma deepens, temperature declines, and the pulse grows weaker, there should be no waiting even for reaction, for hæmorrhage still continues, and there is no hope save in reaching and checking it at its source. In many cases of recognized or suspected epidural hæmorrhage, no question of operation will arise, as with reaction symptoms progressively improve till recovery is complete; and in many others death is too obviously imminent to admit the possibility of a successful issue. It is in the considerable remnant of cases in which symptoms of severity are stationary till reaction, and then with perhaps some hesitating improvement remain undecided for a time, that there may be legitimate doubt as to the propriety or to the time of interference. Even then the more positive the diagnosis of comparatively uncomplicated epidural hæmorrhage, the shorter the period of justifiable indecision. If with the exclusion of serious cerebral lesion symptoms indicate progressive hæmorrhage, the time for inaction has passed. No absolute rules of conduct can be formulated. The necessity of interference in each case, and the time, must be decided by the judgment and experience of the surgeon.

Conditions change when serious injuries affect the parts below the dura-mater. The question of operation then becomes more complicated, whether considered as an abstract proposition or in its relation to individual cases. It cannot be too

often repeated, that while a simple opening of the cranial cavity is devoid of danger, the invasion of the subdural space is never without it; in the one sepsis, always an appreciable possibility, is ordinarily remediable, and hæmorrhage controllable within the limit of safety; in the other, cerebritis with cerebral hernia is a frequent and often fatal complication, and the further loss of blood becomes a matter of perhaps the most serious moment. Shock is absent or trivial when the cranial wall alone is wounded, but is an essential element of danger when the cerebral substance is exposed and subjected to examination. Subdural operation in every case in which it fails of its purpose involves a danger of shock which greatly adds to the chances of a fatal issue of the original traumatism.

In a subdural operation for a meningeal lesion these dangers would be minimized; but as a pial hæmorrhage cannot be distinguished by its direct symptoms from one of epidural origin, nor a subarachnoid serous effusion be isolated from its associated cerebral œdema, they are both likely to fail as direct operative indications. A pial hæmorrhage may be suspected from a combination of the symptoms of hæmorrhage with those of a cerebral œdema, but is oftener unsuspected and disclosed only in the course of a search for the epidural lesion; and in a certain proportion of cases may then be adequately and safely relieved. The removal of a subarachnoid serous effusion is probably never the recognized object of operation, though it may be incidentally accomplished. If, however, either one of these conditions may be sufficiently disassociated from cerebral lesion of importance to permit independent consideration, there can be no question of the propriety of operation when indicated by the gravity of symptoms.

The object to be accomplished by operation in cases of epidural or pial hæmorrhage, or of subarachnoid serous effusion, the relief of pressure, is definite and technically practicable.

It is in the class of intracranial injuries which exclusively or largely involves the brain substance that the greatest uncertainty has been felt as to the advisability of operation. It has been due in part to a failure to discriminate between the visceral

lesion and an epidural or pial hæmorrhage, though a contusion of the brain of such severity as to suggest operation should hardly fail of recognition if ordinary care were used in the examination of the case. If the rise in temperature is not in itself deemed pathognomonic, the loss of rectal or vesical control, or some focal symptom, which is characteristic, ought to afford sufficient confirmation. Even in the presence of a hæmorrhage, some one or more of these indications should be in evidence.

Another cause of uncertainty as to the advisability of operation has been a failure to consider what it might reasonably be expected to accomplish. The essential brain lesions of traumatic origin, as already noted, are laceration and limited or diffuse hyperæmia and œdema. Laceration of the motor area may be recognizable either as an essential or as an incidental lesion, and as such is accessible in operation; but in general lacerations are not distinguishable from the general contusion of which they are part, and in the vast majority of cases are situated at the base or in other inaccessible regions of the brain. Persistent paralyses of the extremities from laceration of the motor area may afford legitimate reason for operation, but it is questionable if the result will often justify expectation. The objects to be obtained are primarily the relief of pressure from attendant hæmorrhage, and, secondarily, the exposure of the cerebral wound for aseptic treatment. The occasional instances in which this method of treatment has been adopted have not demonstrated that it adds to the patient's chance of recovery. Limited contusion, possibly indicated by some focal symptom, can never suggest operation, as it is in itself void of danger.

The one cerebral lesion remaining, which probably exists in all intracranial traumatisms, and is the direct cause of death in a large proportion of fatal cases, is a general hyperæmia and œdema, which primarily exerts an intracerebral pressure, coincidentally or secondarily deranges cellular nutrition, and ultimately tends to destroy vital functions.

If any good can come from trephining in such cases it

must be by relieving intracerebral pressure, and thus permitting the reëstablishment of a normal capillary circulation and the restoration of normal nutritive processes. It is obvious that as with epidural hæmorrhages there are many cases which end in spontaneous recovery as there are many others which equally proceed to an inevitable result. In an intermediate class, in which operative interference might be of possible service, there is an indefinite time beyond which circulatory derangements will have already extended to the vital area, or cellular degeneration will have extended to a point beyond repair. It becomes a question, therefore, not only of what possibility of relief there may be in an operative procedure, but in what cases and within what limit of time it may be effective.

The importance of œdema as a definite and often fatal lesion was long unappreciated; and even yet, in the absence of gross hæmorrhages or lacerations, brains on post-mortem inspection are not infrequently pronounced normal when the œdematous condition is most strongly marked. Not longer ago than 1860, Prescott Hewitt,[4] while he more clearly than his predecessors recognized the fact that so-called concussion was really a structural brain injury, and noted the punctate hæmorrhages and extravasations, made no mention of the serous exudation. In later years, when an intracerebral pressure became more generally accepted as a factor in the genesis of symptoms, there was a disposition to regard hyperæmia of the cerebral vessels and subarachnoid serous effusion as the sole causative agents, even while the existence of cerebral œdema was recognized, and writers had formed various theories as to the manner of its production. The neglect of a study of temperature as an essential element in the diagnosis of intracranial lesions may account for the failure to recognize the importance of œdema in the progression of cerebral symptoms.

The limitations of operative treatment in cases of cerebral hyperæmia and œdema are readily deducible from a consideration of the nature of the changes which occur. Simple circula-

tory derangements are capable of spontaneous readjustment; the cerebral vasomotor centres may recover from the primary shock to which they have been subjected, capillary thromboses and extravasations and incidental serous transudation may be reabsorbed, and initial nutritive changes may be checked. The forces of nature may be aided in their progress to recovery by such remedies as will diminish arterial tension, if it exist and equalize the general circulation; but it is not easy to understand how the action of these natural forces can be facilitated by trephining the cranial wall, since the swelling of the viscus from mere dilatation of the vessels is insufficient to seriously compromise the cranial capacity.

Furthermore, the nutritive derangements are not due solely to mechanical cause. The œdema, which follows primary hyperæmia, and is found post-mortem in some degree in all cases, "is mainly the result of an active process in the tissues themselves."[5] It was regarded as a simple serous transudation due to capillary obstruction until experimental researches were made by Cannon,[5] which seem to have demonstrated that it is the result of "internal nutritive changes" "leading to increased osmotic pressure with passage of water into the tissues," "in which processes blood pressure is not a necessary factor." In either case, whether in the order of events the œdema is the result of an active or of a passive process, whether it is the cause or the sequence, cellular nutrition is progressively impaired and function correspondingly inhibited or destroyed.[6]

It follows that with the lapse of time a new condition is to be met in considering the question of operative treatment. In the beginning, the readjustment of the cerebral circulation may be practicable by the removal of external compression, as is often accomplished in the case of epidural hæmorrhages. It would also be theoretically practicable in the case of cerebral contusions up to the time when structural changes progress beyond the reparable stage, provided intracerebral pressure were the sole cause of the cellular degeneration.

There is, however, reason to believe that it is not the vas-

omotor centres alone which suffer from the primary shock of injury, but that nutritive changes may depend upon a similar impression made upon the nerve cells in general, and thus have an independent origin apart from the circulatory disturbance. It is often noted in post-mortem cases in which epidural hæmorrhage has been large, that there is no indication of any considerable vascular change. The brain is neither markedly hyperæmic, anæmic, or œdematous. Death has not been so sudden as to justify a supposition that there has been paralysis of the respiratory centre, nor are the appearances presented such as to suggest a fatal bulbar anæmia gradually induced by the superincumbent pressure. Again, in cases of uncomplicated contusion, as previously stated, vascular change and œdema are often disproportionate to each other, and often disproportionate to the severity of antecedent symptoms. In some instances the vessels are not greatly dilated, and the œdema is in only moderate degree when so-called pressure symptoms have been severe; in others, œdema is excessive when the blood in cerebral circulation has apparently been neither diminished in amount nor impeded in its course. In all these cases pressure alone, whether extracerebral or intracerebral, is inadequate to explain wholly the progress of symptoms to a fatal termination. That supradural or subdural compression may be a contributing or even the efficient agent in the production of symptoms is evident in many instances from the result of elevation of bone or removal of clot. That intracerebral pressure may be a contributing agent in the progress of the nutritive disorders incident to cerebral contusion may be properly assumed, but its pathological improbability as the essential cause is not diminished by the results of trephining in such cases. The writer, therefore, has been led to modify the view previously expressed,[7] that primary shock affects only the cerebral centres of vascular control, and that its ultimate effect depends solely upon anæmia from pressure. He is now more inclined to believe that the primary impression extends to the nerve cells in general, and that, in the severer cases especially, the fatal nutritive changes result in greater part from the general cellu-

lar implication in shock, and not indirectly through the vascular derangement. If this be true, operative procedure even at an early period is still more likely to be ineffective than if pressure alone, which might in some degree be relieved by the opening of the cranium, were the essential pathological condition.

The cases in which primary shock may be supposed to be essentially limited to the vasomotor centres include all those which speedily recover after the transient unconsciousness with slight disturbances of pulse and temperature. Other cases with similarly restricted lesion, in which unconsciousness and elevation of temperature are somewhat more prolonged, will recover with or without surgical methods short of operation; while those in which from the very first, profound vascular disturbance and rapidly progressive anæmia are indicated, can hardly be benefited by so simple a procedure as the removal of an osseous fragment.

In the larger group of cases in which operation may be proposed, and in which the primary shock has been extended to the general mass of cerebral cells, the relief of pressure by opening the cranium becomes, in the light of Cannon's investigations, and of post-mortem observations previously cited, illogical and ineffective. If the origin of the fatally progressive destruction of cells resides in greater part in an inherent defect of nutrition which is the cause and not the result of œdema, no remedy can avail which ignores this pathological fact. As a pressure from cerebral hyperæmia and a resultant œdema is not the essential cause of the cellular disintegration, which is the real source of danger in the class of cases in which operation is oftenest held to be in question, trephining which can only relieve pressure must be useless and worse than useless, because it makes possible new elements of danger.

If it were conceded that the nature of the lesion were such that at some certain time when natural forces had proved to be ineffectual to stay destructive processes, but when they might be still amenable to control by operative relief of intracerebral pressure, it would even then be impossible to fix that time.

There is no positive progression of symptoms by which the order of pathic changes can be estimated. The gravest symptoms—prolonged unconsciousness, high temperatures, and loss of both rectal and vesical control—may precede more or less sudden change for the better and ultimate complete recovery; or a continued moderate elevation of temperature with no urgent symptoms may be followed by complete loss of consciousness, a high range of temperature, and a speedy fatal termination. Prognosis in cases of moderate severity is little more than guess-work. Temperature is the only indication of value, and this, however positive as a diagnostic sign, or reliable in its exacerbations and recessions as a measure of conditions at the moment, cannot be trusted as pointing to the ultimate result until it has passed the dead line of 105°. In the observation of more than one thousand cases of intracranial injury there was no instance of recovery when that degree of temperature was exceeded by even the smallest fraction.

It may be said that practically all cases of cerebral contusion fall into two classes:

1. Those in which symptoms are not urgent, a large majority of which, as experience teaches, recover by the use of ordinary measures of treatment.

2. Those in which symptoms are of alarming severity, a large majority of which, as experience also teaches, are destined to end in death whatever method of treatment may be adopted.

In neither case are exploratory operations justifiable; in neither one will operation be simply nugatory; it will do harm if it does not do good.

An operation for the purpose of establishing diagnosis is, moreover, unnecessary. Careful analysis and study of symptoms will sufficiently determine the nature of intracranial conditions present to enable the surgeon to exercise his judgment in deciding upon the better course of treatment to pursue. The exact extent and location of lesions may be so far indeterminate as to render the result of an operation uncertain, and in

this sense experimental; but this is not to be confounded with exploration.

It is probable that those surgeons who operate early and often will save many cases which would otherwise have sooner recovered, and that those who operate later in cases which have assumed a graver character will lose some which might have recovered if the cranium had been left intact. It has been the hope of surgeons who have been in the way of seeing many cases of intracranial injury that operation might be extended with advantage to these cases of cerebral traumatism, but the present state of our knowledge of the pathic conditions which obtain affords no reason for believing that this hope will be realized.

SUMMARY.

1. Epidural hæmorrhage demands operation in such cases as do not obviously tend to spontaneous recovery, or in which a fatal issue is so imminent as to permit no question.

2. Meningeal contusion, when productive of symptoms, either cannot be diagnosticated from an epidural hæmorrhage, or is indistinguishable from the diffuse cerebral œdema with which it is always associated. A recognized intracranial hæmorrhage may be expected to be of pial origin when associated with cerebral lesions, and will indicate operation when the cerebral lesion is regarded as of minor importance.

3. Cerebral contusion. (a) Limited—no tendency to a fatal termination, and never suggests operation. (b) Diffuse—two classes of cases; in one, a vascular disturbance incapable of self-limitation, not markedly involving the integrity of the cerebral cells, but tending to mechanically destroy their function; in the other, a progressive disintegration of cellular structure, an active process due to chemical changes, which natural forces prove insufficient to restrain. In the first, operation is theoretically indicated; in the second, in view of the origin and nature of the pathic changes, there is no reason to suppose a simple relief of pressure will stay their progress. In neither is it possible to fix the time when operation may so

supplement natural forces and simpler remedial measures as to increase the patient's chances of recovery.

4. Mixed cases—cerebral contusion complicated with pial or epidural hæmorrhage. Operation should depend upon the estimated relative importance of the lesions; and the correctness of this estimate must depend upon the acumen and experience of the surgeon.

NOTE.—Since this paper was written an article has been published by Murray [8] upon the operative treatment of intracranial hæmorrhages. I quite agree with this writer that the "important point for the surgeon is to recognize the presence of intracranial hæmorrhage, and if the symptoms of compression are severe, to immediately relieve the compression, no matter what may be its source"—if practicable. I am also in accord with him in the belief that "the skull should not be opened in every doubtful case." Some of his statistical and other conclusions, however, are not in consonance with my own observations and experience. Those observers who are quoted as stating that over 90 per cent. die under "expectant treatment," have been singularly unfortunate, if by expectant treatment is meant treatment without operation. I find from the record of my own cases of intracranial injury that of 100 cases of most recent occurrence 30 were fatal. In 16 cases resort was had to operation, with 5 deaths. The 25 fatalities in the 84 non-operative cases represent a fraction less than 30 per cent., practically the same percentage as that noted for the whole series. These were all cases of importance taken in chronological order without selection. The operative cases were: Eleven of depressed fracture of the vertex, 1 of pistol shot wound, 3 of hæmorrhage into the basal fossal with cerebral contusion, and 1 in which operation seems to have been done without adequate reason. Seventeen necropsies were made in the cases where death occurred without operation; and an analysis of post-mortem findings in these shows no one in which operation, if practicable, might be supposed to have been of service. These necropsies, therefore, do not, as expressed

by Dr. Murray, "reveal the fact that early resort to trephining would have been followed by success"; nor does it seem from the history of these cases that, "as regards more frequent exploratory operations it would seem that it is clearly indicated."

Again, the record of 300 or more necropsies which have been made in my own cases do not sustain the allegation that "contusion of the brain is not so frequent an accompaniment of subdural hæmorrhage as was formerly supposed." On the contrary it has been almost uniformly present even when the subdural hæmorrhage has been wholly pial. As previously noted, punctate extravasations and even excessive hyperæmia and œdema are frequently overlooked in post-mortem inspections, and the brain pronounced normal in the absence of laceration.

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[4]Holmes, Surgery, 1860.

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[7]Phelps, Traumatic Injuries of the Brain, 1897–1900. New York Medical Journal, January 11, 1902.

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